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LETTERS TO THE EDITOR

We are pleased to receive Letters to the Editor on appropriate subjects. These letters should be submitted in typewritten form, double-spaced, and are not to exceed 2½ pages. When appropriate, we will solicit comments from the original authors. All Letters to the Editor are subject to editing and possible abridgment.

REGIONAL VARIATION OF IMMUNOGLOBULIN DEPOSITS IN NZB/W F1 MICE: A TAIL-END SUGGESTION.

". . .we do fear this body hath a tail/more perilous than the head."
W. Shakespeare
(in Cymbeline iv-2-144)

To the Editor:

We found the article by Drs. Sontheimer and Gilliam (*J Invest Dermatol* 72:25-28, 1979) on the regional variation of subepidermal immunoglobulin (subepi-Ig) deposits in the NZB/W F1 mouse well done and interesting. Of special note was their finding that subepi-Ig was present on the keratinizing, hair-bearing surface of mouse lip, and its complete absence under the mucosal surface. They hypothesize that at the mucosal surface, the DNA antigen becomes "unavailable to react with DNA antibodies at the D-E junction" since the epithelial cells that are shed at the mucosal surface still contain their nuclei and take the DNA with them [1].

Epidermal cytoplasmic antibodies (ECA) have been linked to keratinization and presumably can detect distinctly different cytoplasmic molecules in keratinocytes "programmed" for mucous, parakeratotic or orthokeratotic differentiation. Therefore, the target antigens for ECA seem to be molecules involved in keratinocyte differentiation [2]. Molecules may change their antigenic characteristics depending upon the type and stage of keratinization in the cell. Further, there is some evidence to suggest that in certain situations, intracellular molecules (antigens), other than DNA, may be deposited into the basement membrane zone at the D-E junction [3]. Cytoplasmic proteins and antibodies to these cytoplasmic proteins have also been described in systemic lupus erythematosis [4].

Since ther NZB/W F1 mice were already injected with tritium-labeled thymidine and biopsies taken from the lip, back and abdomen, it would have been interesting if they had obtained, as well, a biopsy from the tail for their direct immunofluorescent studies; for in that location there are alternating bands of parakeratotic and orthokeratotic epidermis [5]. It would have been additionally interesting if they had tested the sloughed epidermal cells from the parakeratotic tail region with ethidium bromide marker for the presence of DNA along with measuring the basal cell labeling indexes of these tail bands that have the same blood supply, same cephalic-caudal position and the same tissue consistency.

Assuming that DNA antigen is sloughed from the parakeratotic tail region and is therefore unavailable at the D-E junction, these additional procedures would have confirmed their results, strengthened their hypothesis and helped to exclude factors such as skin texture, skin thickness, skin glycogen content and tissue water content that could influence the observed differences between mucosal and parakeratotic skin.

If their observations held true for the tail region, but there was no significant parakeratotic sloughing of DNA in the tail, one would assume that the DNA antigen is available at the D-E junction and the observed differences in mucosal and parakeratotic subepi-Ig deposition are due to some other cause.

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REPLY

We wish to thank Drs. Woodley and Pruniéras for their thoughtful and constructive suggestion and reply simply that the tale of the tail has yet to be told.

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CHRONIC TRICHOPHYTON RUBRUM INFECTION AND BLOODGROUPS

To the Editor:

Drs. Young and Roth state in their article (Immunological Cross-reactivity between a Glycoprotein Isolated from Trichophyton mentagrophytes and Human Isoantigen A, 72: 46–51, 1979) that cross-reacting antigens from the mycelial cell wall of *T. mentagrophytes, T. rubrum* and *E. floccosum* and human isoantigen A, may be a factor leading to chronic infection. They suggest that clinical studies on patients with chronic mycotic infections should include bloodgroup typing.

In our series of 25 chronically with *T. rubrum* infected patients, 20 belonged to the Royal Netherlands Navy personnel, so their bloodgroups could easily be traced. This group of patients was defined as follows. (1) A recorded duration of infection of more than 5 yr. (2) Immediate-type skin reactions to trichophytins in all patients, some which had concurrent delayed-type reactions. (3) Absence of solitary delayed type skin reactions to trichophytins. (4) Normal skin response to other recall antigens. (5) Significantly more atopy and (6) significantly more recurrences in a 2-3 year follow-up period, compared to noninfected patients and a group of patients with a acute *T. rubrum* infection (H. Neering and J. C. van der Horst, to be published).

The distribution of the bloodgroups in these 20 patients was: A, 8 (40%); B, 3 (15%); and O, 9 (45%). Rhesus-D pos were: 18 (90%) and Rhesus-D neg were 2 (10%). This distribution is essentially similar to the occurrence of bloodgroups and Rhesus factors in the Netherlands population (A, 41, 8%; B, 8.5%, AB, 3.0%; O, 46.7%, Rhesus-D pos: 84% and Rhesus-D neg: 16%).

We are, therefore, unable to add clinical evidence to the interesting findings in the article mentioned above.

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TYROSINE HYDROXYLATION

To the Editor:

In their article, Drs. Shapiro, Edelstein, Patel, Okun et al [1] published some rather questionable conclusions based on data which might seem convincing to those not familiar with the field, and which I feel need some clarification. In their paper, they claimed their data supported the notion that mammalian tyrosinase (EC 1.14.18.1) was incapable of tyrosine hydroxylation as determined by the tritiated water assay developed by Pomerantz [2]. There are a variety of papers in the literature, some of ours included, which have shown purified mammalian tyrosinase to be capable of tyrosine hydroxylation, not only via the Pomerantz assay, but by several other types of methodologies as well [3–7]. Space does not allow a full critique of the paper—however, the inability of the Shapiro group to confirm this may be due to one or more of the following considerations: